In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 17-1121V

Filed: July 23, 2024

HEATHER ADAMS,	*
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Petitioner,	*
v.	*
	*
SECRETARY OF HEALTH AND HUMAN SERVICES,	*
	*
Respondent.	*

Leah Durant, Esq., Law Offices of Leah V. Durant, PLLC, Washington, DC, for petitioner.
Ilana Greenberg, Esq., U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

Roth, Special Master:

On August 21, 2017, Heather Adams ("Ms. Adams" or "petitioner") filed a petition for compensation pursuant to the National Vaccine Injury Compensation Program.[2] Petitioner alleges that she suffered from Guillain-Barré Syndrome ("GBS") after receiving an influenza ("flu") vaccine on November 8, 2016. *See* Petition, ECF No. 1.

I. Issues to be Decided

The issues to be decided are whether petitioner suffered from GBS, and if so, whether the Table criteria for GBS have been satisfied.

Petitioner argues that she suffered from GBS and has met the requirements of a Table GBS claim. She argues that the onset of her GBS occurred within 3-42 days of vaccination and virtually

[1] Because this Ruling contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned finds that the identified material fits within this definition, such material will be redacted from public access.

[2] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

all of her treating physicians attribute her symptoms to GBS, despite the presence of a few "atypical" features. Motion at 6, ECF No. 56 (citing Pet. Ex. 2 at 59, 109; Pet. Ex. 4 at 52, 54-55; Pet. Ex. 5 at 59; Pet. Ex. 6 at 11-12; Pet. Ex. 8 at 1, 4, 7, 10-11, 13; Pet. Ex. 13 at 1; Pet. Ex. 14 at 1, 5, 9-10).

Respondent argues that petitioner failed to demonstrate by preponderant evidence that she suffers from GBS or from any other neurological injury caused by the flu vaccine. Therefore, the petition should be dismissed. Response at 2, ECF No. 58.

II. Background

A. Guillain-Barré Syndrome

The supporting literature filed by both petitioner and respondent show that Guillain-Barré syndrome ("GBS") is an immune-mediated demyelinating polyradiculoneuropathy with a broad clinical spectrum characterized by different patterns of nerve conduction failure resulting in symmetrical progressive muscle weakness with loss of reflexes. Pet. Ex. 17[3] at 1; *see* Pet. Ex. 21[4] at 1, Resp. Ex. A, Tab 1[5] at 1. "The cardinal clinical features consist of progressive relatively symmetrical weakness, mild sensory symptoms and areflexia", and the cranial and respiratory muscles can be affected as well. *Id*.; *see also* Pet. Ex. 20[6] at 1. GBS is usually preceded by infection or other immune stimulation that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots. Resp. Ex. A, Tab 1[7] at 1. Two-thirds of GBS patients report preceding symptoms of a respiratory or gastrointestinal infection within four weeks of onset of weakness. *Id*. at 2. C-jejuni, cytomegalovirus, Epstein-Barr virus, influenza A virus, mycoplasma pneumonia, and haemophilus influenzae virus have all been associated with GBS. *Id*. "Cases of Guillain-Barre syndrome have also been reported shortly after vaccination with Semple rabies vaccine and various types of influenza A vaccine." *Id*. at 2; Resp. Ex. A, Tab 2[8] at 1-2. In 1976, 100,000 people who were vaccinated for H1N1 influenza A virus developed GBS. Resp. Ex. A, Tab 1[9] at 2.

The key presenting symptom in most patients is rapidly progressive bilateral weakness, with weakness classically described as ascending and starting in the distal lower extremities, "but can start more proximally in the legs or arms." Resp. Ex. A, Tab 1[10] at 5. In about 10% of patients, weakness begins in the arms or facial muscles. Resp. Ex. A, Tab 2[11] at 2. In more than 80% of patients, weakness is accompanied by paresthesias in the hands and feet, but sensory abnormalities on examination are frequently mild. *Id*. EMG/NCS are valuable for confirming a diagnosis of GBS

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[3] Forsberg et al., *Residual disability 10 years after falling ill in Guillain-Barré syndrome: A prospective follow-up study*, 10 J. NEUROLOGICAL SCI. 1016 (2012), filed as "Pet. Ex. 17."
[4] Marcel P. J. Garssen et al., *Nerve conduction studies in relation to residual fatigue in Guillain-Barré syndrome*, 253 J. NEUROLOGY 851 (2006), filed as "Pet. Ex. 21."
[5] Hugh J. Willison et al., *Guillain-Barré syndrome*, 388 LANCET 717 (2016), filed as "Resp. Ex. A, Tab 1."
[6] Tiina Rekand et al., *Fatigue, pain and muscle weakness are frequent after Guillain-Barré and poliomyelitis*, 256 J. NEUROLOGY 349 (2009), filed as "Pet. Ex. 20."
[7] Willison et al., *supra* note 5.
[8] Francine J. Vriesendorp, *Guillain-Barré syndrome in adults: Clinical features and diagnosis*, UPTODATE (Oct. 8, 2019), https://uptodate.com, filed as "Resp. Ex. A, Tab 2."
[9] Willison et al., *supra* note 5.
[10] *Id*.
[11] Vriesendorp, *supra* note 8.

but is not required for diagnostic or treatment purposes. EMG/NCS can also provide some information regarding prognosis and help with classifying variants. *Id*. at 8. GBS is classically known for albuminocytological dissociation, which is the combination of a normal CSF white blood cell count and an elevated CSF protein level. CSF is important in diagnosing GBS and excluding alternative diagnoses. Resp. Ex. A, Tab 2[12]at 7. Albuminocytological dissociation is present in up to 66% of patients one week after onset of symptoms and in greater than 75% of patients three weeks after onset. *Id*. at 3, 7. "A normal CSF protein is found in one-third to one-half of patients when tested earlier than one week after symptom onset and therefore does not exclude the diagnosis of GBS." *Id*. at 7. Features such as marked persistent asymmetry of weakness and bowel and bladder dysfunction at onset make a GBS diagnosis doubtful. *Id*. at 9. "…CSF is examined mainly to exclude disorders that are associated with pleocytosis, instead of seeking confirmation of the diagnosis Guillain-Barre syndrome by demonstrating an increased protein concentration." Resp. Ex. A, Tab 3[13] at 10.

The acute progression of limb weakness, often with sensory and cranial nerve involvement one to two weeks after immune stimulation, proceeds to its peak clinical deficit in two to four weeks:

> When patients present with rapidly progressive paralysis, the diagnosis of Guillain-Barré syndrome needs to be made as soon as possible. Although establishment of the diagnosis in typical cases is usually straightforward, there are many clinical and investigative components to consider, especially in atypical cases. The diagnosis is largely based on clinical patterns because diagnostic biomarkers are not available for most variants of the syndrome.

Resp. Ex. A, Tab 1[14] at 1. IVIG or plasma exchange has proven beneficial and is crucial, especially in patients with rapidly progressive weakness. *Id*.

GBS was initially thought to be a single disorder but is now recognized as a heterogeneous syndrome with several variant forms, each with distinguishing clinical pathophysiologic and pathologic features. *See* Resp. Ex. A, Tab 2[15] at 4-7.

Pain, fatigue, and muscle weakness are the most common complaints following GBS. Pet. Ex. 20[16] at 4. Most patients show relatively good neurological recovery after GBS, but severe fatigue and endurance intolerance can be disabling long-term effects. Pet. Ex. 21[17] at 1. The underlying pathophysiology of fatigue in patients who have recovered from GBS is unknown. *Id*. Three-quarters of all patients reported fatigue that affects their quality of life, and fatigue is more prevalent in females and patients over the age of 50. Pet. Ex. 18[18] at 1, 3. There was no significant

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[12] Vriesendorp, *supra* note 8.
[13] Christiaan Fokke et al., *Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria*, 137 BRAIN 33 (2014), filed as "Resp. Ex. A, Tab 3."
[14] Willison et al., *supra* note 5.
[15] Vriesendorp, *supra* note 8.
[16] Rekand et al, *supra* note 6.
[17] Garssen et al., *supra* note 4.
[18] M.P.J. Garssen et al., *Residual fatigue is independent of antecedent events and disease severity in Guillain-Barré syndrome*, 253 J. NEUROLOGY 1143 (2006), filed as "Pet. Ex. 18."

relationship found between functional disability at nadir, clinical variables, and antecedent events or infections. *Id*.

In one study, patients across all GBS variants reported pain, and most described it as moderate to severe. One-third of patients reported pain as an initial symptom occurring up to two weeks before the onset of weakness, and two-thirds of patients reported pain during the acute phase, within the first three weeks after onset of their condition. 38% of patients had pain within the first year. Pet. Ex. 19[19] at 3. Anxiety and depression were also reported in the first year following GBS and chronic inflammatory demyelinating polyneuropathy ("CIDP"), with physical activity noted to bring significant relief. *Id*. at 4.

B. Procedural History

The petition was filed on August 21, 2017, and assigned to the Special Processing Unit ("SPU"). Petition, ECF No. 1. On September 5, 2017, petitioner filed medical records, an affidavit, and a statement of completion. Petitioner's Exhibits ("Pet. Ex.") 1-7, ECF Nos. 7-8.

An initial status conference was held on October 11, 2017. Respondent filed a status report identifying records that appeared outstanding on April 9, 2018, and petitioner filed the outstanding records and a statement of completion on May 15, 2018. ECF No. 15; Pet. Ex. 8-10, ECF No. 18-19. Respondent then requested additional records on June 14, 2018, which were filed with a statement of completion on July 19, 2018. ECF No. 20; Pet. Ex. 11-13, ECF Nos. 22-23.

On October 18, 2018, respondent advised by status report that he was willing to engage in settlement discussions. ECF No. 28. The parties engaged in settlement negotiations for approximately six months. ECF Nos. 29-35. On May 10, 2019, petitioner filed a status report advising that despite respondent's willingness to negotiate on a litigative risk basis, respondent had not yet filed a Rule 4(c) Report with his view of the case, and petitioner's counsel felt there was insufficient information to advise her client about respondent's settlement offer. ECF No. 36.

Respondent then filed his Rule 4(c) Report on June 27, 2019, recommending against compensation because petitioner's symptoms and diagnosis "do not fit squarely within the QAI for [GBS]" and there was a lack of confirmatory diagnostic testing. ECF No. 37; ECF No. 38 at 7. The case was transferred from SPU to the undersigned on July 16, 2019. ECF Nos. 39-40.

At an initial status conference on November 1, 2019, petitioner advised that she would like to proceed with the filing of expert reports. ECF No. 42.

Petitioner filed expert reports from Dr. Hoke with corresponding medical literature. Pet. Ex. 15-21, ECF Nos. 46-47; Pet. Ex. 22, ECF No. 52; Pet. Ex. 23, ECF No. 55. Respondent filed expert reports and literature from Dr. Chaudhry. Respondent's Exhibit ("Resp. Ex.") A-B, ECF No. 49; Resp. Ex. C, ECF No. 53.

[19] Ingemar S.J. Merkies & Bernd C. Kieseier, *Fatigue, Pain, Anxiety and Depression in Guillain-Barré Syndrome and Chronic Inflammatory Demyelinating Polyradiculoneuropathy*, 75 EUR. NEUROLOGY 199 (2016), filed as "Pet. Ex. 19."

On May 23, 2021, petitioner filed a Motion for Ruling on the Record. Motion, ECF No. 56. Respondent filed a response on August 30, 2021. Response, ECF No. 58. Petitioner filed a reply on October 25, 2021. Reply, ECF No. 61. Thereafter, respondent filed a response to petitioner's reply on February 3, 2022. Supp. Response, ECF No. 64.

Having determined that the parties have had a full and fair opportunity to present their cases, it is appropriate to resolve this issue without a hearing. See Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *Kreizenbeck v. Sec'y of Health & Human Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020) (noting that "special masters must determine that the record is comprehensive and fully developed before ruling on the record."). Accordingly, this matter is now ripe for resolution.

C. Factual History

1. Medical History Prior to the Flu Vaccine

Petitioner's past medical history includes low vitamin D, headaches, fatigue, daytime somnolence, lethargy, and incidences of fainting. Pet. Ex. 2 at 5, 7, 11, 17, 31-32, 39. Petitioner was prescribed oral phentermine, phendimetrazine, and B12 HCG injections for weight loss. *See generally* Pet. Ex. 9. Petitioner also suffered from polycystic ovarian syndrome ("PCOS") with irregular periods for which she was prescribed Metformin and Provera. Pet. Ex. 10 at 2.

On October 11, 2016, petitioner presented to her PCP with a host of complaints including headaches, hair loss, inability to lose weight, and feeling "always tired." Pet. Ex. 2 at 46. She was assessed with fatigue and labs were ordered. *Id*.

Petitioner returned to the PCP on October 31, 2016, with a sore throat for about a week, cough, wheezing, and chest tightness when breathing. She had no fever, body aches, or headache and the examination was normal. She was diagnosed with bronchitis, prescribed azithromycin, and advised to rest, hydrate, and use antipyretics and albuterol as needed. Pet. Ex. 2 at 48.

On November 8, 2016, petitioner filled out a flu screening form that she was healthy and received the subject influenza vaccine in her right arm. Pet. Ex. 2 at 50; Pet. Ex. 1 at 1.

2. Medical History Following the Flu Vaccine

On December 7, 2016, petitioner presented to the PCP with one month of head and chest congestion, nasal drainage, dry cough, and bilateral earache. She had previously been prescribed a Z-pack and felt better but became ill again. She did not have a headache, fever, limb weakness, myalgias, arthralgias, or gait or balance issues. Pet. Ex. 2 at 51. Neurological examination was normal. She was diagnosed with acute sinusitis and prescribed Bactrim and cough medicine. *Id.* at 52-53.

Four days later, on December 11, 2016, petitioner presented to Jane Phillips Medical Center Emergency Room ("ER") with weakness and inability to use her hands or straighten her fingers. She reported gradual onset of weakness, fatigue, and abdominal pain with nausea and vomiting. Pet. Ex. 5 at 95. Examination found no focal neurological deficits, intact cranial nerves,

and equal bilateral motor strength with a normal sensory examination. Patellar and Achilles reflexes were 2/5. Gait and speech were normal. Pet. Ex. 5 at 97. Laboratory results showed elevated white blood count ("WBC"), creatinine, glucose, and neutrophils. *Id*. at 98. A chest x-ray was clear, and a head CT was negative. *Id*. at 98-99, 112, 113. She reported onset of symptoms on Friday, a recent sinus infection, and a flu shot one month ago. Pet. Ex. 2 at 66. The record documented upper extremity weakness but no signs of GBS at this time. Pet. Ex. 5 at 100, 102. She was administered IV fluids and discharged home to follow up with her PCP. *Id*. at 107. She needed assistance dressing and reported worsening weakness since arrival to the ER. *Id*. She also reported difficulty urinating but refused a straight catheter and requested a pregnancy test. *Id*. at 118.

Petitioner presented to her PCP the next day for follow-up of generalized motor weakness in her upper and lower extremities. An upper respiratory infection over the past month was noted, treated with azithromycin and Bactrim with no fever, chills, body aches, cough, congestion, shortness of breath, or wheezing. Pet. Ex. 2 at 55. Petitioner reported some dizziness and numbness and tingling in her hands and feet on Friday, and weakness in her arms and legs on Saturday when she awoke. Her symptoms worsened yesterday. *Id*. She went to the ER, had bloodwork, x-rays, and a head CT and was told it may be a reaction to Bactrim. *Id*. She now complained of a severe inability to move her legs and arms with numbness and tingling in her hands and feet. There was no facial weakness, dizziness, vertigo, blurred vision, speech problems, incontinence, or pain, but she was exhausted. *Id*. There was weakness on examination but sensory was intact. The impression was questionable GBS. She was sent by private car to St. John Medical Center ("SJMC") in Tulsa. *Id*. at 56.

Upon admission to SJMC, petitioner reported arm and leg weakness that began with finger numbness and grip strength issues on Saturday and progressed since. Pet Ex. 4 at 33. She was worse today and could not walk. She denied incontinence, recent travel, injury, fever, or chills. She reported taking Bactrim last Wednesday for an upper respiratory infection and feeling dizzy a few days later. *Id*. The assessment was progressive weakness, first in the upper extremities then in the lower extremities. She was unable to ambulate or bear weight. She had motor and sensory deficits of unclear etiology in the upper and lower extremities. *Id*. at 34. An MRI of the cervical spine with and without contrast showed "[p]resumed subtle artifact like hyperintense signal of the mid cervical cord on the sagittal postcontrast series…Otherwise normal appearing spinal cord." *Id*. at 91-92.

Dr. Edgar provided a neurological consultation. Pet. Ex. 4 at 31. Petitioner reported mild malaise on Friday, and numbness in her fingers and weakness in her hands and feet when she awoke on Saturday that worsened throughout the day and was much worse on Sunday. She went to the ER but was discharged. She presented to her PCP on Monday unable to stand and was taken by car to SJMC. Her weakness had progressed since admission, and she was now unable to "get her hands up to her face." *Id*. She had mild lower back pain earlier that mostly went away. "She does not having (sic) tingling or numbness right now," headache, stiff neck, nausea, or vomiting. *Id*. Dr. Edgar's impression was "[p]robably Guillain-Barre syndrome, although given the lack of auxiliary symptom/signs (hyponatremia, low back pain, acral paresthesias) I think viral poliomyelitis needs to remain a consideration." *Id*. He added that a CSF profile would probably clarify the diagnosis, with albuminocytological dissociation meaning GBS, and lymphocytic

pleocytosis meaning viral infection, but it can take a week for CSF to become abnormal in some GBS cases. *Id*. Dr. Edgar further noted that petitioner's course was aggressive, and he anticipated she would need intubation. He discussed with her an expected worsening over the next several days, nadir within one to two weeks, a plateau at nadir for one to two weeks, then slow recovery over a few months. He also discussed IVIG that would modulate her course, possibly minimizing deficits at nadir, hastening onset of recovery, and likely speeding up recovery once it began. *Id*.

Petitioner's CSF testing showed rare white blood cells but no epithelial cells or bacteria. Pet. Ex. 4 at 40-41. Labs were normal. *Id*. at 47. On December 14, 2016, Dr. Edgar documented that petitioner was slightly weaker than yesterday with normal CSF, "as it often is this early on, but [the] goal was to rule out poliomyelitis which has been accomplished." IVIG was started. *Id*. at 52. Dr. Edgar's assessment was "Guillain-Barre syndrome, more rapidly progressive than average so I still expect need for intubation relatively soon, but it does seem that rapidity has slowed in the past 24h." *Id*.

The following day, Dr. Edgar noted that petitioner had regained "just a bit of proximal arm movement compared to yesterday," with no new issues or worsening problems. There was moderate to severe upper and lower extremity weakness, areflexia, mild facial diplegia, mild to moderate neck flexion weakness, and no dysarthria. Pet. Ex. 4 at 54. His impression was GBS. He noted that she may already be approaching nadir, which would be "unusually quick but fortunate" to avoid intubation. He expected a plateau over the next few days followed by slow improvement, but she was not "out of the woods yet." *Id*. IVIG was to continue, with inpatient rehabilitation needed unless her convalescent phase was more rapid than typical. *Id*.

On December 16, 2016, Dr. Edgar noted that petitioner had a bit more strength and was already at the plateau phase of GBS, with some remaining risk of worsening. Her last IVIG was scheduled for the following day. Pet. Ex. 4 at 55.

Petitioner was noted to be more comfortable by December 17, 2016, with the strength in her arms and legs improving daily. Pet. Ex. 4 at 59. She was able to grip weakly with her hands, lift her arms 6-8 inches off the bed, and move her legs more. The diagnosis remained GBS, improving slowly, with need for inpatient rehabilitation at discharge. *Id*.

By December 19, 2016, Dr. Edgar noted that petitioner was getting stronger every day. She had no swallowing or breathing problems. She was regaining strength in her hands, legs, and feet. The facial diplegia was gone. There was fair improvement in the upper extremities and good improvement in the lower extremities. Pet. Ex. 4 at 62. Dr. Edgar expected her return to premorbid functional baseline within three months, if not sooner, with weekly strides made over the next several months. His impression remained GBS. *Id*.

Petitioner was willing to go to inpatient rehabilitation to "get back to her life." Pet. Ex. 4 at 64. Her condition was documented at that time as GBS with functional quadriplegia and improving muscle strength in the upper and lower extremities after five days of IVIG. *Id*. She was discharged on December 21, 2016. *Id*. at 65. The discharge summary[20] included a principal

[20] The discharge summary was not contained in the record from SJMC but was found in the record from petitioner's PCP. *See* Pet. Ex. 2 at 80.

diagnosis of "[a]cute Guillain-Barre syndrome with respiratory failure."[21] Pet Ex. 2 at 80. The record noted that petitioner "was found to have Guillain-Barre syndrome by EMG and was transferred to the intensive care unit due to impending respiratory failure."[22] Pet. Ex. 2 at 80. Dr. Edgar was consulted, the patient received a full course of IVIG, stabilized, and subsequently improved. She was discharged to Kaiser Rehab. *Id*.

The admission record for Hillcrest Medical Center/Kaiser Medical Center ("Hillcrest"), an in-patient rehabilitation facility, documented a 29-year-old mother of a 3-year-old living with her fiancé and working full-time. Pet. Ex. 3 at 57. She had an onset of lower extremity weakness that "kept basically ascending into her trunk and numbness in her fingers" that began on December 12, 2016. She was able to walk into the ER but unable to walk out. She was admitted to SJMC, diagnosed with GBS, and received IVIG. *Id*. She had an upper respiratory infection and was taking Bactrim three days prior to the onset of weakness. She was now stable and ready to start rehabilitation. *Id*. at 51, 57. She denied vision changes, problems with coughing, chewing, swallowing or choking, shortness of breath, or chest pain, but her appetite was poor. She reported that initially she had some bladder control issues but had good control now. She denied numbness, tingling, or pain. *Id*. at 58. Neuromuscular testing showed reduced strength in her upper and lower body: areflexic in the right upper extremity with no deep tendon reflexes on the left; lower extremity strength was fairly symmetrical with no reflexes found in either knee. Her clinical presentation was noted to be consistent with her preadmission assessment. *Id*. She was to receive physical therapy ("PT") and occupational therapy ("OT") at a minimum of three hours a day with an estimated stay of ten to 14 days. *Id*. at 59.

Petitioner improved and within days needed only minimal to moderate assistance for bed mobility, minimal assistance for transfers, supervision to minimal assistance for gait, and was ambulating 50-100 feet using a rolling walker. Pet. Ex. 3 at 55. She requested to be discharged on December 23, 2016, even though her goals were not met. She was determined to be home for Christmas. A social worker was not present, so therapies needed to be set up after she was home. She was examined and noted to have considerable OT deficits but was ambulatory and considered safe for discharge. Pet. Ex. 3 at 6, 15-20, 52-53.

On December 29, 2016, petitioner presented to her PCP for follow up. She was improved, gaining strength daily, and walking better but still "waddling." PT and OT appointments were scheduled for the following week. Pet. Ex. 2 at 109. Influenza vaccine was listed as an allergy in the record. *Id*. The assessment was "Guillain Barre syndrome - recovering." *Id*. at 110.

Petitioner received outpatient OT and PT at Jane Phillips Medical Center Physical Medicine Center. Pet. Ex. 5 at 11. She had an OT evaluation on January 4, 2017 for a diagnosis of GBS. She complained of decreased strength in both wrists and all fingers but was independent in performing self-care activities. *Id*. at 32. She had difficulty with heavier lifting activities. She denied pain, swelling, numbness, or tingling in either upper extremity with normal sensation. Her active range of motion was normal, but the right upper extremity did lag slightly behind the left due to weakness. *Id*. She had made "a fairly quick recovery" from GBS and was ambulating

[21] Petitioner never suffered respiratory failure, although precautions were taken due to the rapid progression of her condition.

[22] There is no record of EMG testing being done during petitioner's hospitalization.

without assistive devices. She had some upper extremity weakness, especially in the right shoulder and both hands. *Id*. at 33. A plan and goals were set for OT twice per week for eight weeks. *Id*. at 33-34.

Petitioner had a PT evaluation on January 4, 2017. The history of her GBS was documented. She reported that she never lost sensation but lost the function of her upper and lower extremities. Pet. Ex. 5 at 45. The record notes that "[t]he patient is doing amazingly well considering the short period of time since the onset. She just basically has general strength issues." *Id*. Her range of motion was within normal limits for all four extremities. She complained of her lower back and legs aching at night, but pain was otherwise not an issue. *Id*. She was independent without assistive devices and demonstrated good gait speed and heel-toe gait pattern but had some slight imbalance laterally. She denied any sensation deficits. *Id*. at 46. She was noted to be motivated and presented "following an onset of Guillain-Barre syndrome after receiving a flu shot." She had made excellent progress to date and would benefit from strength and balance PT two times per week for four to eight weeks. *Id*.

Petitioner presented to her PCP on January 19, 2017, requesting permission to return to work. She was attending PT and OT and gaining strength and was much improved but still slightly weak. She was able to return to work with no restrictions and directed to finish OT and PT as planned. Pet. Ex. 2 at 125-26.

Petitioner completed eight OT visits between January 4, 2017 and February 2, 2017, with good improvement in upper extremity strength. She still had weakness in her right hand, especially with grip and pinch strengths. She reported little functional ability impairment and was tolerating work and home activities. She was educated on home exercises and discharged from OT. Pet. Ex. 5 at 38.

Petitioner was discharged from PT on February 16, 2017, after five visits between January 6, 2017 and January 23, 2017. She had made good progress and returned to work. An official re-evaluation could not be done because petitioner did not complete her PT sessions. She was compliant with home exercises and had no pain. She was primarily working on high-level strengthening. Pet. Ex. 5 at 44.

At a six-month follow-up examination on May 10, 2017, her history of GBS was documented, in the "fall 2016 after flu vaccine; hospitalized and went through PT. Still having intermittent weakness and fatigue". Pet. Ex. 6 at 2. She worked on a computer, was having difficulty typing, and becoming frustrated and depressed. She gained weight and lost interest in things she was normally interested in. She reported trying to go out and walk, but her legs felt heavy and at times dragged, her knees buckled, and she felt she had no strength. She had no numbness, tingling, dizziness, or vision changes. Her gait was normal and motor and sensory were intact. *Id*. Prozac was started. Her fatigue and weakness were noted as possible residuals from GBS, but labs were ordered to rule out other causes. *Id*. at 3. Her vitamin D was still low, her glucose was high, and her thyroid function was abnormal. *Id*. at 1.

Petitioner returned for a Prozac check on June 7, 2017, with improved mood but some irritability. Pet. Ex. 6 at 5. The assessment was depression. Prozac was increased. *Id*. at 5-6.

Petitioner returned two weeks later with improved mood but one week of neck pain that was worse after work. On examination, she had decreased range of motion of her neck, but no numbness, tingling, or weakness in her arms. Pet. Ex. 6 at 8.

At a July 3, 2017 visit, petitioner reported persistent weakness, paresthesia, and muscle fatigue, particularly in her hands when she types. Her fingers would not "work right" and it felt like her knees gave out at times when walking. Pet. Ex. 6 at 11. Her depression had improved with Prozac, but she was still gaining weight and not as active as she once was. She was bothered by ongoing fatigue and weakness in her upper and lower extremities. Bloodwork was negative for anemia or thyroid disease, but her vitamin D was still low despite taking ergocalciferol and getting sun exposure. The assessment was depression improved on Prozac and weakness, fatigue, and paresthesia likely the residual effects of GBS. She was referred to Dr. Edgar for follow up. *Id*. at 11-12.

Petitioner returned to Dr. Edgar on July 19, 2017, who noted her recovery from GBS with some remaining but not disabling symptoms. Pet. Ex. 13 at 1. Dr. Edgar advised that it may take another six months for the paresthesias to resolve and that it was not uncommon for GBS patients to have some minor sensory complaints and intolerance with prolonged activity. *Id*. He wrote, "[s]he believes this occurred as a complication of influenza vaccination, and that is possible although not provable. This does not mean that she cannot have other vaccinations in the future, but it would probably be prudent to avoid influenza vaccinations." *Id*. The examination was normal for motor, coordination, gait, and reflexes. *Id*.

In October 2017, petitioner presented to her PCP with Family and Medical Leave Act ("FMLA") paperwork. Pet. Ex. 8 at 1. She was struggling with weakness, paresthesia, and muscle fatigue in her lower extremities, with weakness in her hands and fingers when typing. She felt her legs would give out. She had ongoing fatigue. She had some bad days, but her depression was overall controlled with Prozac. *Id*. PT was offered again to improve strength, but she declined because she did not want to miss time from work. Yoga was suggested. *Id*. at 2.

Petitioner returned to the PCP on February 9, 2018, reporting one month of muscle and nerve pain in her lower extremities with leg "jumping" affecting her sleep, weakness, and fatigue. Pet. Ex. 8 at 4. Prozac was not working. She was more irritable, stressed, and unable to find joy in things she normally did. She was assessed with depression, muscle fatigue that was possibly residual from GBS, and restless leg syndrome. She was prescribed a trial of gabapentin and a dose increase of her antidepressant. *Id*.

At a March 2018 follow up visit, petitioner reported no improvement in depression with increased medication. Pet. Ex. 8 at 7. She still had numbness, tingling, and weakness in her lower extremities at times, but not daily. She had GBS a year and a half ago with residual weakness but had not seen the neurologist since July 2017. She was not taking the gabapentin because it made her sleepy and did not help. *Id*. She had normal gait, strength, range of motion, and sensory to light touch. Prozac was changed to Zoloft. She was referred to neurology. *Id*. at 7-8.

Petitioner had improvement with Zoloft, but still had increased irritability and feelings of hopelessness some days. She was waiting for an appointment with Dr. Edgar. Pet. Ex. 8 at 10.

Petitioner continued to report lower leg numbness, tingling, and weakness on May 4, 2018. Pet. Ex. 8 at 13. She was encouraged to do lower leg exercises and to consider PT if there was no improvement. *Id*.

Petitioner presented to Dr. Lajara-Nanson, a neurologist, on May 24, 2018. He noted Dr. Edgar's diagnosis of GBS. Pet. Ex. 11 at 1. Petitioner reported bilateral lower extremity weakness, general fatigue, and burning and tingling in her hands at night. She reported a flu shot two weeks before the onset of numbness in her fingertips, followed by profound weakness the following day. She was then hospitalized and "was paralyzed from the neck down but did not require intubation." She was treated with IVIG. *Id*. She reported no pain at that time but began to have hand cramping, intermittent weakness, and burning pain in her hands and the lateral aspects of both thighs a few weeks later. *Id*. After a neurological examination, Dr. Lajara-Nanson wrote:

> I find no clear evidence that [petitioner] suffered from Guillain-Barre Syndrome. Spinal fluid studies at the time of diagnosis were normal and she has no motor weakness, no distal sensory loss in the upper and lower limbs. In addition, her reflexes are intact; these usually do not return after Guillain-Barre; certainly not one that would lead to quadriplegia and persistent symptoms.

Pet. Ex. 11 at 3. Dr. Lajara-Nanson believed petitioner had meralgia paresthetica of the lower limbs, not uncommon in obese individuals and a potential precursor to diabetes, but "a peculiar residual for GBS." *Id*. He prescribed Lyrica. He found that she had carpal tunnel syndrome in both wrists and ordered splints and EMG/NCS testing of both the upper and lower extremities. "This diagnosis makes more sense with her reported intermittent symptoms and daily typing." *Id*.

Petitioner returned to her PCP on May 29, 2018. She reported that the neurologist prescribed Lyrica and recommended EMG testing. Pet. Ex. 12 at 1. She had not picked up the Lyrica yet. She recounted her visit to the neurologist and his belief that she did not have symptoms of GBS but rather was overweight. *Id*. The PCP increased her Zoloft and recommended she pursue EMG testing and take the Lyrica to see if it helped. She requested a different neurologist. *Id*. at 2.

No records of EMG testing were filed. The next medical record filed was from April 24, 2019, at which time petitioner reported having had a sleep study at Bailey Medical Center and being diagnosed with obstructive sleep apnea.[23] Pet. Ex. 14 at 1. She was using a CPAP machine with benefit and slightly increased energy. She continued to complain of weakness with numbness and tingling in her upper and lower extremities, some days worse than others. She was taking gabapentin with improvement on some days. She had not seen a neurologist in some time. She was referred to a neurologist and directed to continue gabapentin and anxiety medication. *Id.* at 1-2.

On June 26, 2019, petitioner presented to the PCP after falling out of the shower that morning when her knee "gave away." Pet. Ex. 14 at 5. She now had right hip and leg pain she rated as 7/10. The PCP cautioned her to take it slow due to her bilateral lower extremity weakness. Icing and ibuprofen were recommended. *Id*. at 5-6.

[23] It does not appear that petitioner filed the records from this sleep study.

Petitioner's last medical visit filed was with her PCP in September 2019. Pet. Ex. 14 at 9. She continued to suffer from bilateral upper and lower extremity weakness, numbness, and tingling with flares that come and go, some days worse than others. She was taking gabapentin, which was effective half of the time. She tried an increased dose, but it made her too tired. *Id*. She was compliant with the CPAP and was taking Zoloft, which was working well. She was having irregular, painful, heavy periods and had stopped taking Metformin due to stomach upset. *Id*.

3. Petitioner's Affidavit

Petitioner affirmed receipt of a flu vaccine as required by her job. Pet. Ex. 7 at 1. Within two weeks of the vaccination, she felt muscle fatigue and was tired all the time. *Id*.

Petitioner affirmed that while shopping on December 9, 2016, she noticed that she had lost grip strength and could not control her hands or maneuver a shopping cart around the store. Pet. Ex. 7 at 1. The next day, she started to lose the ability to stand unassisted. She presented to the ER and was advised it could be GBS, but the doctor was not sure because it started with her hands and stated, "it should wear off." He suggested she contact her PCP the next morning if she had concerns. By the end of the visit, she was unable to stand without assistance, and her husband had to carry her into the house. *Id*.

Petitioner affirmed that by the next morning, she could not move anything other than her head from side to side. Pet. Ex. 7 at 1. She went to the PCP, who called in another doctor for consult, and was directed to go to SJMC in Tulsa immediately because they believed she may have GBS. She was admitted to SJMC. *Id*.

Petitioner affirmed she was told she was being moved to the ICU because they believed she had GBS, and "the next course this syndrome could take would be to take away my ability to breathe unassisted." Pet. Ex. 7 at 1. Once in the ICU, petitioner had a spinal tap to rule out multiple sclerosis ("MS"). She was moved to the recovery floor after five days in ICU and discharged three days later. An EMG was not done while she was at SJMC. *Id*. at 2.

According to petitioner, she was transferred to Kaiser Rehab Center for physical therapy on December 21, 2016, and discharged to outpatient physical therapy closer to her home on December 23, 2016. She completed occupational and physical therapy. Pet. Ex. 7 at 2.

Petitioner affirmed that she still experiences fatigue, muscle spasms, and loss of strength. Her knees buckle with extended activity and her hands cramp or spasm, making typing difficult and forcing her to stop what she is doing to do hand exercises from the occupational therapist. Pet. Ex. 7 at 2.

Petitioner affirmed requiring treatment for depression due to the financial strain of the overwhelming medical bills incurred. Her personal life has also been affected because she cannot do the things that she used to do, such as playing on the floor with her son or picking him up. Pet. Ex. 7 at 2.

III. The Experts

A. Petitioner's Expert, Dr. Ahmet Hoke

1. Qualifications

Dr. Hoke is a neuromuscular specialist whose practice focuses on peripheral nerve disorders in an academic setting. He is a professor of neurology and neuroscience at Johns Hopkins University School of Medicine, Director of the Neuromuscular Division, Director of the Neuromuscular Fellowship, and Co-Director of Neuromuscular Histopathology Laboratory. He is on the medical staff of the neurology and pathology departments at Johns Hopkins Hospital. Additionally, he conducts translational laboratory research on the pathogenesis of peripheral neuropathies and nerve regeneration. Pet. Ex. 15 at 1; *see* Pet. Ex. 16.

2. Dr. Hoke's Opinion

Dr. Hoke detailed petitioner's medical history. Pet. Ex. 15 at 1-8. Dr. Hoke's opinion is that petitioner had GBS and demonstrated ascending weakness, paresthesia, evolving areflexia, and ataxia, all "classical features of GBS. *Id*. at 8.

Dr. Hoke added that while other diagnoses were considered, all were ruled out by testing and CSF testing ruled out infectious etiology. An MRI of the spine and the lack of bowel and bladder problems ruled out transverse myelitis ("TM"). Pet. Ex. 15 at 8. Dr. Hoke addressed the "artifact" on the MRI, noting that petitioner had no upper motor neuron signs throughout her hospitalization, which also argues against a TM diagnosis. Pet. Ex. 23 at 2; Pet. Ex. 2 at 58. While Dr. Chaudhry raised various possible alternative diagnoses including CIDP, TM, and MS, the required testing to advance those diagnoses was never performed. Pet. Ex. 23 at 2. Dr. Hoke acknowledged that at petitioner had preserved reflexes at her initial presentation on December 11, 2016, but she had numbness and tingling and was areflexic when examined by a neurologist on December 12, 2016. *Id*.; Pet. Ex. 2 at 55-56. This rapid loss of reflexes fits a presentation of GBS, not TM or MS. Pet. Ex. 23 at 2. Further, if she had CIDP, she would not have had a normal examination with intact reflexes 18 months later. *Id*.; Pet. Ex. 11.

According to Dr. Hoke, all GBS patients do not present with the same symptoms, and the fact that an EMG was not performed does not mean petitioner did not have GBS: "[i]t would have been nice to have it as a confirmatory test but her treating physicians felt that the evidence was sufficient to make a diagnosis of GBS after ruling out other alternative diagnoses." Pet. Ex. 23 at 1-2.

Dr. Hoke discussed petitioner's upper respiratory symptoms and sinus congestion "at some point prior to the onset of her GBS", noting that none of her medical providers attributed her GBS to an infectious cause and no record specified the existence of infection. Pet. Ex. 22 at 1; Pet. Ex. 23 at 1. References to respiratory symptoms in the record were simply comments associated with temporal events preceding the onset of weakness. Pet. Ex. 23 at 1. Further, the exclusionary criteria for a Table GBS injury "makes no reference to ill-defined generalized infectious processes" such as a sinus infection, congestion, or upper respiratory infection. Pet. Ex. 22 at 2.

Dr. Hoke addressed Dr. Lajara-Nanson's office visit 18 months after petitioner's GBS diagnosis, in which he assessed her with carpal tunnel syndrome and meralgia paresthetica. Dr. Hoke acknowledges that Dr. Lajara-Nanson's assessment may have been accurate on the day he examined her, but it does not detract from her GBS diagnosis a year and a half prior. He noted that "[c]arpal tunnel syndrome is a very common condition and meralgia paresthetica is not uncommon in overweight individuals." Pet. Ex. 15 at 7-8.

Further, Dr. Hoke relied on *Garssen*, *Rekand*, *Forsberg*, and *Merkies & Keiseier* to show that GBS patients can have persistent fatigue, muscle weakness, and paresthesia even after full recovery on nerve conduction studies. The literature "clearly demonstrate[s] the incorrect rationale" of Dr. Lajara-Nanson that petitioner did not suffer from GBS because she had good recovery of reflexes and strength. Pet. Ex. 15 at 8; Pet. Ex. 18[24]; Pet. Ex. 21[25]; Pet. Ex. 20[26]; Pet. Ex. 17[27]; Pet. Ex. 19.[28]

Dr. Hoke opined that, "[petitioner] had classical features of GBS…and her relatively rapid recovery from quadriparesis was partly due to her age and rapid initiation of IVIG treatment before she reached the nadir of her illness." Pet. Ex. 15 at 9. Residual fatigue is not uncommon in GBS patients who have full recovery. *Id*. Dr. Hoke concluded that petitioner suffered GBS within 3-42 days of her flu vaccine. *Id*.

B. Respondent's Expert, Dr. Vinay Chaudhry

1. Qualifications

Dr. Chaudhry is a neurologist who specializes in neuromuscular diseases, including the management of peripheral neuropathies. He is also an expert in electrodiagnostic studies as they relate to neuromuscular diseases. Resp. Ex. A at 1. Dr. Chaudhry is board certified in neurology, neuromuscular diseases, electrodiagnostic medicine, and clinical neurophysiology. He has an active clinical practice and evaluates over 2000 patients a year, mostly related to peripheral nerve disease. He is involved in clinical research, reviews articles for over 35 different journals, and has been on the editorial board of three journals. He has directly mentored over 50 fellows and several hundred medical students and neurology residents. He is considered an expert in the evaluation and treatment of patients with peripheral neuropathies, including GBS and CIDP. Resp. Ex. A at 1; Resp. Ex. B.

2. Opinion

Dr. Chaudhry summarized petitioner's medical history. Resp. Ex. A at 2-8. He described GBS as an acquired disease of the peripheral nervous system characterized by rapidly progressing ascending paralysis with parethesias. Resp. Ex. A at 8. Areflexia and involvement of the cranial

[24] Garssen et al., *supra* note 18.
[25] Garssen et al., *supra* note 4.
[26] Rekand et al., *supra* note 6.
[27] Forsberg et al., *supra* note 3.
[28] Merkies & Kieseier, *supra* note 19.

nerves, diaphragm, and autonomic nervous system are common. *Id*. Nerve conduction studies generally show demyelinating electrophysiology and CSF evaluation will show albuminocytological dissociation. GBS is a monophasic illness with a good prognosis for recovery, and both IVIG and plasma exchange are effective treatments. *Id*.

Dr. Chaudhry agreed that petitioner had a rapid onset of limb weakness and absent reflexes consistent with GBS, but her "other features" raised doubt about the diagnosis. Resp. Ex. A at 8. Dr. Chaudhry elaborated on these "other features", including weakness that started in the upper extremities rather than in an ascending pattern; marked asymmetry in abduction between the right and left shoulder and right and left hip; normal sensory examination despite the presence of paresthesias; and lack of bladder control. *Id*. Dr. Chaudhry added that her subsequent course was also atypical for GBS, with continuing symptoms and intermittent flares of paresthesias and weakness up to three years after her acute symptoms. He wrote that GBS is not associated with relapses and worsening weakness, and these fluctuations raise the possibility of acute onset CIDP. *Id*. at 9. Still further, Dr. Chaudhry added that petitioner did not have albuminocytological dissociation on CSF, which is typical for GBS. EMG and nerve conduction studies were not done during the acute phase of her illness or in follow-up, despite petitioner's persistent symptoms of paresthesias and weakness. The Brighton criteria requires EMG testing to diagnose GBS. Resp. Ex. A at 9; Resp. Ex. C at 3.

Dr. Chaudhry contends that petitioner's treating physicians failed to explore alternative diagnoses. Resp. Ex. A at 9. They failed to rule out TM in light of her complaint of bladder issues and MRI results showing subtle artifact-like hyperintense signal in the mid-cervical cord. Repeat imaging was not done. *Id*. at 12; Pet. Ex. 2 at 112. Her asymmetry on right and left shoulder abduction was suggestive of C5 myeloradiculopathy. Resp. Ex. A at 12. An MRI of the brain was not done to rule out lesions on the brain like those on the C-spine, since TM can be the initial event that correlates with MS. Blood tests such as serum anti-aquaporin-4-IgG autoantibodies and anti-myelin oligodendrocyte glycoprotein antibodies were not performed and may have clarified her condition. *Id*.; Resp. Ex. C at 3. Petitioner's continuing intermittent weakness and giving way of her knees may have been a feature of a spinal cord lesion with depressed reflexes during the acute stage of spinal cord disease. *Id*.

Dr. Chaudhry argues that petitioner has CIDP due to the fluctuation of her symptoms up to three years later. Resp. Ex. A at 10; Pet. Ex. 8 at 13; Resp. Ex. A, Tab 6.[29] Like petitioner, CIDP patients do not require a ventilator. Like petitioner, CIDP patients have significantly less cranial nerve dysfunction. Resp. Ex. A at 10; Resp. Ex. A, Tab 7.[30] Like petitioner, one-third of CIDP patients have a relapsing-remitting course, more common in younger patients. Resp. Ex. A at 10; Resp. Ex. A, Tab 9.[31] Dr. Chaudhry argues that nerve conduction studies, which were not done here, were important to confirm a diagnosis of CIDP. Resp. Ex. A at 10. Fatigue is a common

[29] Liselotte Ruts et al., *Distinguishing acute-onset CIDP from Guillain-Barré syndrome with treatment related fluctuations*, 65 NEUROLOGY 138 (2005), filed as "Resp. Ex. A, Tab 6."
[30] L. Ruts et al., *Distinguishing acute-onset CIDP from fluctuating Guillain-Barré syndrome: A prospective study*, 74 NEUROLOGY 1680 (2010), filed as "Resp. Ex. A, Tab 7."
[31] Richard A. Lewis, *Chronic inflammatory demyelinating polyneuropathy: Etiology, clinical features, and diagnosis*, UPTODATE (Aug. 29, 2018), https://uptodate.com, filed as "Resp. Ex. A, Tab 9."

symptom of CIDP and can be a presenting feature. Petitioner experienced fatigue that predated the onset of her acute weakness. *Id*.

According to Dr. Chaudhry, Dr. Hoke failed to address the symptoms that raised doubts about petitioner's GBS diagnosis and failed to discuss the unclear etiology of her condition and the other diagnoses raised by her treating physicians including paresthesias, TM, myelopathy, and proximal myopathy. Resp. Ex. C at 1-2, 5; Pet. Ex. 2 at 61-66. Dr. Chaudhry referred to Dr. Lajara-Nanson's assessment to support his opinion that petitioner did not have GBS because her CSF testing was normal, she did not have motor weakness or distal sensory loss in the upper and lower limbs, and her reflexes were intact, which does not occur with GBS. Resp. Ex. C at 2; Pet. Ex. 11 at 3.

Dr. Chaudhry raised additional possible diagnoses based on petitioner's medical history, including idiopathic intracranial hypertension to explain her headaches; hormonal irregularities to explain her hair loss, tiredness, and unexplained weight gain; and myopathies associated with cardiac arrhythmias to explain her episodes of syncope in 2016 that were thought to be vasovagal. Resp. Ex. A at 10. Finally, Dr. Chaudhry added that petitioner has been diagnosed with obstructive sleep apnea which is usually associated with fatigue, tiredness, and headaches. *Id*. at 11.

Dr. Chaudhry found it difficult to address causation because petitioner's diagnosis was uncertain and EMG testing was never done. Resp. Ex. A at 11. Further, GBS is a post-infectious immune disorder, with two thirds of patients having diarrhea or an upper respiratory illness several weeks before the onset of symptoms. *Id*.; *see* Resp. Ex. A, Tab 1[32]; Resp. Ex. A, Tab 2[33]; Resp. Ex. A, Tab 3.[34] GBS has been associated with C. jejuni infection in 25-50% of GBS patients, cytomegalovirus, Epstein-Barr virus, influenza A virus, mycoplasma pneumoniae, Haemophilus influenza, hepatitis E, Zika, and chikungunya. GBS has also been reported following vaccination for rabies and rabies and influenza A. Resp. Ex. A at 11; Resp. Ex. A, Tab 1[35] at 2.

According to Dr. Chaudhry, petitioner had three possible infections prior to onset of weakness and paresthesias. The first was an upper respiratory infection on October 31, 2016, five to six weeks prior to onset, with sore throat, cough, wheezing, and chest tightness. Resp. Ex. A at 11; Pet. Ex. 2 at 48. The second was another upper respiratory infection on December 7, 2016, three days prior to onset, with head and chest congestion, earaches, nasal drainage, and cough. Resp. Ex. A at 11; Pet. Ex. 2 at 51. Third, bloodwork done on December 13, 2016 was positive for EBVG and EBNUCL AB, though Dr. Chaudhry conceded that these findings do not suggest acute infection, but noted that Epstein-Barr Virus is a known antecedent to GBS. Resp. Ex. A at 11; Resp. Ex. C at 4; Pet. Ex. 2 at 48, 51, 74. In Dr. Chaudhry's opinion, if petitioner had GBS, it was caused by the immediate antecedent upper respiratory infection. He added that Dr. Hoke ignored these infections. Resp. Ex. A at 11-13; Pet. Ex. 2 at 61-66, 68, 82-83, 88-93, 111; Pet. Ex. 4 at 47. Dr. Chaudhry contends that the failure to identify a specific infection does not mean an infection was not the cause of her GBS. Resp. Ex. C at 3.

[32] Willison et al., *supra* note 5.
[33] Vriesendorp, *supra* note 8.
[34] Fokke et al., *supra* note 13.
[35] Willison et al., *supra* note 5.

Succinctly, Dr. Chaudhry opines that petitioner's GBS diagnosis is unclear because she had ongoing intermittent paresthesias and weakness after the acute episode, her MRI was abnormal, her CSF testing was normal, no EMG was performed to confirm the diagnosis, her weakness started in her upper body and was not ascending, and she did not have an ataxic gait. Resp. Ex. A at 12. Other possible diagnoses including TM, MS, early myelopathy, proximal myopathy, meralgia paresthetica, carpal tunnel syndrome, and restless leg syndrome were raised by her treating physicians, and Dr. Lajara-Nanson also questioned the GBS diagnosis in May 2018. *Id*. Therefore, it is uncertain that petitioner had GBS, and even assuming that she did, the evidence demonstrates that preceding infections may have been the antecedent trigger. The flu vaccine was not likely the cause of her symptoms. *Id*. at 14.

IV. Discussion

A. Standard for Adjudication

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. 42 U.S.C. § 300aa-11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health & Human Servs*., 165 F.3d 1344, 1352 (Fed. Cir. 1999). Petitioners are not required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs*., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes"). Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs*., 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing 42 U.S.C. § 300aa-13(a)(1)(B)).

Petitioner alleged only a Table claim for GBS following the flu vaccine. Therefore, the *Althen* requirements need not be addressed.

In enacting the Vaccine Act that included the Vaccine Injury Table, Congress turned the maxim "post hoc ergo propter hoc" on its head. *Shalala v. Whitecotton*, 514 U.S. 268, 270 (1995). When a petitioner establishes that she "sustained . . . any illness . . . set forth in the Vaccine Injury Table in association with [a] vaccine . . . and the first symptom . . . of the onset . . . occurred within the time period after the vaccine administration set forth in the Vaccine Injury Table," the petitioner gains a rebuttable presumption that the vaccination caused the injury. *Whitecotton*, 514 U.S. 270-71; 42 U.S.C. § 300aa–11(c)(1)(C)(i). The Secretary may rebut this prima facie showing

by proving that the illness was in fact caused by "factors unrelated to the administration of the vaccine." 42 U.S.C. § 300aa–13(a)(1)(B); *accord Whitecotton*, 514 U.S. at 270-71.

In trying to establish that a factor unrelated to the vaccination caused the injury, the Secretary's burden is a "preponderance of the evidence." *Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1150 (Fed. Cir. 2007). A "preponderance of the evidence" is not the same as scientific certainty. *Bunting v. Sec'y of Health & Human Servs*., 931 F.2d 867, 873 (Fed. Cir. 1991).

In the present case, for petitioner to qualify for a Vaccine Injury Table presumption of causation, a preponderance of the evidence must establish that the initial symptom or manifestation of onset of her GBS occurred not less than three days and not more than 42 days after the vaccination. 42 C.F.R. § 100.3(a). Petitioner must also establish that she satisfies the criteria for a Table GBS injury in the Act's qualifications and aids to interpretation ("QAI"), set forth at 42 C.F.R. § 100.3(c)(15). The QAI specify that:

> GBS is an acute monophasic peripheral neuropathy that encompasses a spectrum of four clinicopathological subtypes . . . the interval between the first appearance of symptoms and the nadir of weakness is between 12 hours and 28 days. This is followed in all subtypes by a clinical plateau with stabilization at the nadir of symptoms, or subsequent improvement without significant relapse. Treatment related fluctuations in all subtypes of GBS can occur within 9 weeks of GBS symptom onset and recurrence of symptoms after this time-frame would not be consistent with GBS.

42 C.F.R. § 100.3(c)(15)(i).

Moreover, to "qualify as any subtype of GBS, there must not be a more likely alternative diagnosis for the weakness." 42 C.F.R. § 100.3(c)(15)(v). In particular, the "[e]xclusionary criteria for the diagnosis of all subtypes of GBS include the ultimate diagnosis of any of the [listed] conditions," and the list is not exhaustive. *See* 42 C.F.R. § 100.3(c)(15)(vi); *see also* 42 U.S.C. § 300aa-13(a)(1). The Act also requires petitioner to have "suffered the residual effects or complications of such illness, disability, injury, or condition for more than 6 months after the administration of the vaccine." 42 U.S.C. § 300aa-11(c)(1)(D)(i).

Although this decision discusses some but not all the literature in detail, I have reviewed and considered all the medical records and literature submitted in this matter. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); *Simanski v. Sec'y of Health & Human Servs.*, 115 Fed. Cl. 407, 436 (2014) ("a Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

B. Analysis

After review of the record, including the medical records, expert reports, literature, and the parties' briefs, and in accordance with the applicable legal standards, I find that petitioner's diagnosis is GBS, and she has proven by preponderant evidence that she meets the Table criteria for GBS following a flu vaccination. Accordingly, I find petitioner entitled to compensation based on the following.

1. Treating Physicians Diagnosed Petitioner with and Treated Her for GBS

Petitioner was healthy when she received the influenza vaccine on November 8, 2016. Pet. Ex. 1 at 1; Pet. Ex. 2 at 50.

On December 11, 2016, she presented to the ER with two days of gradual onset of weakness, fatigue, abdominal pain, nausea, vomiting, and inability to open her hands. Pet. Ex. 5 at 95. Examination was normal but patellar and Achilles reflexes were 2/5. *Id*. at 97. She reportedly grew weaker during her ER visit with difficulty urinating. *Id*. at 118. She was administered fluids and discharged home to follow up with her PCP. *Id*. at 107.

The following day, petitioner presented to her PCP with generalized weakness in her upper and lower extremities. She reported dizziness, numbness, and tingling in her hands and feet on Friday, and weakness in her arms and legs when she woke on Saturday which worsened yesterday. She was unable to walk and went to the ER. Her PCP noted weakness on examination but no sensory deficits. Pet. Ex. 2 at 55; Pet. Ex. 5 at 97. The impression was "questionable GBS", and she was sent to the hospital by private vehicle. Pet. Ex. 2 at 55-56.

Upon admission to the hospital, a detailed history of the onset of her symptoms was taken. Pet. Ex. 4 at 33. She denied bladder or bowel incontinence, reported taking Bactrim for a URI last Wednesday, and was unable to bear weight or ambulate. Her weakness had progressed, and she now had sensory deficits in both her upper and lower extremities on examination. *Id*. at 33-34. An MRI showed "[p]resumed subtle artifact like hyperintense signal of the mid cervical cord on the sagittal postcontrast series…Otherwise normal appearing spinal cord." *Id*. at 91-92.

Dr. Edgar conducted a neurological examination on December 13, 2016, with the impression being "[p]robably" GBS. He ordered a lumbar puncture to rule out viral infection. Pet. Ex. 4 at 31, 40-41. On the same day, Dr. Calvert noted progressive weakness with an assessment of "apparent guillan (sic) barre syndrome". *Id*. at 47. He also noted petitioner "has had a recent upper respiratory infection treated with bactrim (for a couple days), and the flu shot 3 weeks ago." *Id*. Dr. Calvert transferred petitioner to the ICU and IVIG was initiated. *Id*.

Dr. Edgar noted petitioner to be slightly weaker with breath support mildly reduced, and some neck flexion weakness on December 14, 2016. He documented that the CSF testing was normal "as it often is this early on, but goal was to rule out poliomyelitis which has been accomplished." Pet. Ex. 4 at 52. His diagnosis was "Guillain-Barre syndrome, more rapidly progressive than average so I still expect need for intubation relatively soon, but it does seem that rapidity has slowed in the past 24h". *Id*. On December 15, 2016, Dr. Edgar noted that he felt she

was reaching nadir more quickly than usual which was fortunate if she could avoid intubation, but she was not "out of the woods." *Id*. at 54. Throughout her stay, Dr. Edgar's diagnosis remained GBS. Pet. Ex. 4 at 55, 60, 62.

Dr. Lawler also assessed petitioner with "Guillan (sic) Barre Syndrome – ivig 12/13-12/17" on December 17, 2016, and noted she was improving slowly. Pet. Ex. 4 at 59-60. On December 19, 2016, following completion of IVIG treatment, Dr. Lawler documented, "Guillan (sic) barre syndrome – with functional quadriplegia…" *Id*. at 63.

On December 20, 2016, Dr. Wenger documented petitioner as having GBS with improving muscle strength in the upper and lower extremities bilaterally following IVIG. Pet. Ex. 4 at 64-65. Petitioner was discharged to inpatient rehabilitation with a diagnosis of GBS. Pet. Ex. 2 at 80.

Petitioner's subsequent visits all document a diagnosis of GBS. Pet. Ex. 2 at 110, 118, 121, 125; Pet. Ex. 6 at 2, 11; Pet. Ex. 5 at 11, 32, 45; Pet. Ex. 13. The only exception was Dr. Lajara-Nanson, who saw petitioner 18 months after her GBS diagnosis. *See* Pet. Ex. 11. Dr. Lajara-Nanson disagreed with Dr. Edgar's diagnosis of GBS, and based on his own examination, assessed petitioner's ongoing lower leg complaints as meralgia paresthetica due to her weight and her upper extremity complaints as carpal tunnel syndrome from typing. He suggested EMG/NCS studies and Lyrica. Pet. Ex. 11 at 3.

A host of potential diagnoses were considered at the time of petitioner's admission to the hospital, including paresthesias, GBS, TM, myelopathy, and proximal myopathy. *See* Pet. Ex. 2 at 66. However, petitioner's treating physicians all agreed her diagnosis was GBS after CSF testing ruled out an infectious cause and an MRI found a "[p]resumed subtle artifact like hyperintense signal of the mid cervical cord on the sagittal postcontrast series…Otherwise normal appearing spinal cord." Pet. Ex. 4 at 91-92. Petitioner was treated with IVIG for GBS. None of her physicians ordered additional testing for or diagnosed her with anything but GBS. In the three years following her diagnosis, petitioner suffered from residual symptoms attributed to her GBS. *See* Pet. Ex. 6 at 2, 11-12; Pet. Ex. 8 at 1-2, 4; Pet. Ex. 13 at 1.

The opinions of and diagnosis made by petitioner's treating physicians are entitled to great weight. *Capizzano v. Sec'y of Health & Human Services,* 440 F. 3d. 1317, 1320, 1325-26 (Fed. Cir. 2006). After review of the medical records and the opinions of petitioner's treating physicians, I find that petitioner's diagnosis was GBS.

2. Petitioner's Onset was Between 3 and 42 days After Vaccination

For a flu GBS claim to be on-Table, the first manifestation must be between 3 and 42 days after receipt of vaccination. 42 C.F.R. §100.3(a)(XIV)(D). It is undisputed that petitioner received the subject vaccination on November 8, 2016, and presented to the hospital on December 11, 2016 with the onset of weakness a day earlier, placing petitioner's onset within the 3 to 42 days period. Thus, I find that onset fell within the Table definition for GBS and flu vaccine.

3. Petitioner Satisfies the Qualification and Aids to Interpretation for a Table GBS Claim following Flu Vaccination

A Table claim for GBS requires the following:

A. Bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in weak limbs;
B. A monophasic illness pattern;
C. An interval between onset and nadir of weakness between 12 hours and 28 days;
D. Subsequent clinical plateau (the clinical plateau leads to either stabilization at the nadir of symptoms, or subsequent improvement without subsequent relapse; however, death may occur without a clinical plateau); and,
E. The absence of an identified more likely alternative diagnosis.

42 C.F.R. §100.3(c)(15)(ii).

Supportive but not required evidence for a GBS diagnosis includes EMG findings consistent with GBS or an elevation of CSF protein with a total CSF white blood cell count below 50 cells per microliter. However, both CSF and EMG testing are frequently normal in the first week of illness in otherwise typical cases of GBS. 42 C.F.R. §100.3(c)(15)(iv).

Exclusionary criteria for the diagnosis of GBS include the ultimate diagnosis of conditions including CIDP, MS, and more. 42 C.F.R. §100.3(c)(15)(vi).

Petitioner's medical records, the opinions of her treating physicians, and the opinion and supportive evidence outlined by Dr. Hoke provide persuasive evidence that petitioner has met these criteria.

i. Bilateral Flaccid Limb Weakness and Decreased or Absent Deep Tendon Reflexes in Weak Limbs

Petitioner's medical records reveal that while petitioner's development of symptoms was initially atypical, she ultimately suffered from motor and sensory deficits of her upper and lower extremities, areflexia, mild to moderate neck flexion weakness, and mild facial diplegia, satisfying the criteria for bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in the weak limbs. Pet. Ex. 5 at 97; Pet. Ex. 2 at 55-56; Pet. Ex. 4 at 34, 52, 54.

When petitioner first presented on December 11, 2016, there were no focal neurological deficits, her cranial nerves were intact, and there were normal motor and sensory findings bilaterally, but her patellar and Achilles reflexes were 2/5. Pet. Ex. 5 at 97. The next day, she presented with worsening symptoms, including weakness and numbness and tingling in her hands and feet, but no sensory involvement. There was no facial weakness, dizziness, vertigo, blurred vision, speech problems, incontinence, or pain. Pet. Ex. 2 at 55-56. However, she was sent directly to the hospital and upon arrival had motor and sensory deficits in the upper and lower extremities with areflexia and mild facial diplegia in the days that followed. Pet. Ex. 2 at 55-56; Pet. Ex. 4 at 33-34, 52, 54.

Dr. Hoke pointed out that although petitioner had preserved reflexes on December 11, 2016, she had numbness and tingling on December 12 and was areflexic when examined by Dr. Edgar. Pet. Ex. 15 at 2-3; Pet. Ex. 2 at 55, 59-60. Dr. Hoke opined that this rapid loss of reflexes fits a "typical" pattern of GBS. Pet. Ex. 23 at 2.

Dr. Chaudhry agreed that petitioner had a rapid onset of limb weakness and absent reflexes consistent with GBS but argued that her other features raised doubt about the diagnosis. Resp. Ex. A at 8.

While petitioner's initial presentation may have been unusual, her symptoms progressed quickly to motor and sensory deficits in the upper and lower extremities, with areflexia among other deficits. Pet. Ex. 5 at 95, 97; Pet. Ex. 2 at 55-56; Pet. Ex. 4 at 33-34, 52, 54.

Petitioner's medical records show that she suffered from weakness, paresthesia, numbness and tingling of the upper and lower extremities, inability to bear weight, and areflexia leading to a diagnosis of GBS, thus meeting the criteria for bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in the weak limbs. I find that petitioner has satisfied this requirement.

ii. A Monophasic Illness Pattern

Petitioner was admitted to the hospital on December 12, 2016 with rapidly progressing GBS. IVIG was started to treat GBS following an MRI and lumbar puncture. Pet. Ex. 4 at 31. On December 15, 2016, Dr. Edgar noted that petitioner had regained "just a bit of proximal arm movement compared with yesterday" with no new issues or worsening problems. Pet. Ex. 4 at 54. Dr. Edgar noted moderate to severe upper and lower extremity weakness, areflexia, mild facial diplegia, and mild to moderate neck flexion weakness. He felt she may already be reaching nadir, which would be "unusually quick but fortunate." Further, he expected a plateau over the next few days followed by slow improvement. *Id*. The next day, Dr. Edgar noted that petitioner was getting stronger daily. *Id*. at 59. By December 19, 2016, Dr. Edgar noted fair improvement in her upper extremities and good improvement in her lower extremities. *Id*. at 62. Upon discharge from the hospital to a rehabilitation facility on December 21, 2016, petitioner was noted to have improvement in muscle strength in the upper and lower extremities after five days of IVIG and had stabilized. *Id*. at 64-65; Pet. Ex. 2 at 80.

Petitioner made further improvement as an inpatient at a rehabilitation facility. Pet. Ex. 3 at 6, 15-20, 52-53, 188. After her discharge from rehabilitation, petitioner continued to suffer from weakness that was "intermittent", "residual", "ongoing", and considered a residual symptom of GBS by her treating physicians. Pet. Ex. 6 at 2-3, 11-12; Pet. Ex. 8 at 1, 7, 10, 13; Pet. Ex. 14 at 1, 5-6, 9-10. On March 5, 2018, her PCP wrote that it had been "1.5 years with residual weakness" after her GBS diagnosis. Pet. Ex. 8 at 7.

Dr. Hoke highlighted one of Dr. Edgar's records, which explained that it is "common for a GBS patient to end up with some relatively minor sensory complaints and intolerance of prolonged activity." Pet. Ex. 15 at 6-7 (citing Pet. Ex. 13). Dr. Hoke filed literature in support of this opinion, showing that persistent symptoms of fatigue, muscle weakness, and paresthesia are

not uncommon amongst individuals who have "fully" recovered from GBS. Pet. Ex. 15 at 8; Pet Ex. 17[36]; Pet. Ex. 18[37]; Pet. Ex. 19[38]; Pet. Ex. 20[39]; Pet. Ex. 21.[40]

Dr. Chaudhry disagrees that petitioner had a monophasic illness and contends that petitioner's "continual symptoms with intermittent flares" are symptoms of CIDP, not GBS. Resp. Ex. A at 9; *see* Pet. Ex. 6 at 2-3, 11-12; Pet. Ex. 8 at 1, 7, 10; Pet. Ex. 14 at 1, 5-6, 9-10.

Based on the record, I find that petitioner's illness stabilized after five days of IVIG treatment as documented by Dr. Edgar. Pet. Ex. 4 at 31, 55. Petitioner then continued to improve with therapy and returned to her daily activities but continued to suffer from ongoing residual symptoms. There is no reference in the medical records to any significant deterioration in symptoms or concerns that petitioner experienced any relapses. She was consistently noted to have residual complaints associated with her GBS. I therefore find that petitioner's illness was monophasic, and she has met this requirement.

iii. An Interval Between Onset and Nadir of Weakness Between 12 Hours and 28 Days

There is no dispute about the onset of petitioner's symptoms. Petitioner presented to the ER on December 11, 2016, with two days of symptoms which continued to develop and worsen until she was admitted to the hospital and quickly diagnosed GBS, "more rapidly progressive than average". IVIG was initiated with improvement noted. Pet. Ex. 4 at 45-46, 52-54. By December 15, 2016, Dr. Edgar noted that petitioner had regained "just a bit of proximal arm movement compared with yesterday" with no new issues or worsening problems. *Id*. at 54. Despite Dr. Edgar documenting moderate to severe upper and lower extremity weakness, areflexia, mild facial diplegia, and mild to moderate neck flexion weakness, he also noted that petitioner may already be reaching nadir, which would be "unusually quick but fortunate." Further, he expected a plateau over the next few days followed by slow improvement. *Id*. By December 16, 2016, Dr. Edgar noted that petitioner had a bit more strength and was already at the plateau phase of GBS. Her last IVIG treatment was scheduled for the following day. *Id*. at 55.

The assessment of petitioner's treating neurologist, Dr. Edgar, was that she reached nadir during the five-day course of IVIG treatment, within the 12 hours and 28-day requirement. Therefore, petitioner has satisfied this requirement.

iv. Subsequent Clinical Plateau Leading to Stabilization at the Nadir of Symptoms or Subsequent Improvement Without Significant Relapse

As detailed above, petitioner reached nadir within the five-day period during which she was receiving IVIG treatment. She was then discharged to an inpatient rehabilitation center until

[36] Forsberg et al., *supra* note 3.
[37] Garssen et al., *supra* note 18.
[38] Merkies & Kieseier, *supra* note 19.
[39] Rekand et al., *supra* note 6.
[40] Garssen et al., *supra* note 4.

just before Christmas, when she was discharged home. On December 29, 2016, petitioner presented to her PCP, reporting much improvement, and walking better though still "waddling." Pet. Ex. 2 at 109. On January 4, 2017, she was noted to have made "a fairly quick recovery" and was ambulating without assistive devices. She continued to exhibit some upper extremity weakness, especially in the right shoulder and both hands. Pet. Ex. 5 at 33. At a physical therapy evaluation on the same date, petitioner was noted as "doing amazingly well considering the short period of time since the onset. She just basically has general strength issues." *Id*. at 45.

At her visit to her PCP on January 19, 2017, petitioner was improved but still slightly weak. She was able to return to work with no restrictions but needed to complete her therapies. Pet. Ex. 2 at 125-26. Petitioner completed her OT and PT between January 4, 2017 and February 2, 2017, with good improvement in her upper extremity strength, but she still had weakness in her right hand especially with grip and pinch strengths. She reported little functional ability impairment and had returned to work with good endurance tolerating work and home activities. Pet. Ex. 5 at 38-39, 44.

Since that time, petitioner has remained stable with continued complaints of ongoing weakness in her upper and lower extremities and fatigue considered by her treating physicians to be residual symptoms of her GBS. Pet. Ex. 6 at 2-3, 11-12; Pet. Ex. 13; Pet. Ex. 8 at 1-2, 4, 7, 13; Pet. Ex. 14 at 9-10.

Dr. Chaudhry referred to petitioner's ongoing symptoms as flares indicative of CIDP rather than GBS. Resp. Ex. A at 10. Dr. Hoke maintained that these symptoms were consistent with residual complaints following GBS even in those who have fully recovered, and petitioner's full recovery with intact reflexes would be inconsistent with CIDP. Pet. Ex. 15 at 8; Pet. Ex. 23 at 2.

I find that petitioner has met this criterion as well. Petitioner reached nadir and plateaued following five days of IVIG treatment. She improved but continued to have ongoing residual symptoms, none of which were considered by her treating physicians to be relapses or deteriorations, and none required further IVIG treatment. Pet. Ex. 6 at 3, 11-12; Pet. Ex. 13; Pet. Ex. 14 at 5-6. Petitioner has satisfied this requirement.

v. The Absence of an Identified More Likely Diagnosis

Respondent's expert opines that there are a host of alternative diagnoses and causes for petitioner's conditions. Resp. Ex. A at 9-11.

Dr. Chaudhry described GBS as an acquired disease of the peripheral nervous system characterized by rapidly progressing ascending paralysis with parethesias. Resp. Ex. A at 8. Areflexia and involvement of the cranial nerves, diaphragm, and autonomic nervous system are common. *Id*. Nerve conduction studies generally show demyelinating electrophysiology and CSF evaluation will show albuminocytological dissociation. GBS is a monophasic illness with a good prognosis for recovery, and both IVIG and plasma exchange are effective treatments. *Id*.

Dr Chaudhry agreed that petitioner had a rapid onset of limb weakness and absent reflexes consistent with GBS, but he argued that her other features raised doubt about the diagnosis being

GBS. Resp. Ex. A at 8. He took issue with petitioner's initial symptoms of weakness in her upper extremities rather than ascending upward; the asymmetry in abduction between the right and left shoulder and right and left hip; intact sensory initially despite the presence paresthesia; and lack of bladder control not normally seen with GBS. *Id*. Further, he referred to her continuing flares of paresthesias and weakness up to three years after her acute symptoms as atypical of GBS, raising the possibility that she suffers from CIDP. *Id*. at 9. Dr. Chaudhry also took issue with petitioner's CSF being normal and that no EMG and nerve conduction studies were done during the acute phase of her illness or in follow-up, despite petitioner's persistent symptoms of paresthesias and weakness. Resp. Ex. A at 9; Resp. Ex. C at 3.

Dr. Chaudhry argues that alternative diagnoses were not explored. Resp. Ex. A at 9. He submits that the artifact on MRI and petitioner's bladder issues were an indication of TM, which should have prompted additional testing that was not done. Resp. Ex. A at 12. An MRI of the brain should have been done to rule out lesions similar to what was seen on the cervical spine, because TM is an initial event that correlates with MS. *Id*. at 9, 12. The asymmetry between right and left shoulder abduction was suggestive of C5 myeloradiculopathy. *Id*. at 12. Petitioner's continuing intermittent weakness and giving way of her knees may be a feature of a spinal cord lesion, with depressed reflexes during the acute stage of spinal cord disease. *Id.*at 9. Blood tests such as serum anti-aquaporin-4-IgG autoantibodies and anti-myelin oligodendrocyte glycoprotein antibodies were not performed and may have clarified her condition. *Id*.; Resp. Ex. C at 3.

In Dr. Chaudhry's opinion, it is more likely that petitioner has CIDP in light of the symptom fluctuations she experienced up to three years later. Resp. Ex. A at 10; Resp. Ex. A, Tab 6[41]; Pet. Ex. 8 at 13. Additionally, like petitioner, CIDP patients do not require a ventilator and have significantly less cranial nerve dysfunction. Resp. Ex. A at 10; Resp. Ex. A, Tab 7.[42] Further, like petitioner, one-third of CIDP patients have a relapsing-remitting course, and this is more common in younger patients. Resp. Ex. A at 10; Resp. Ex. A, Tab 9.[43] Nerve conduction studies, which are important in confirming CIDP, were never done. Resp. Ex. A at 10. Fatigue is a common symptom of CIDP and can be a presenting feature, and petitioner complained of fatigue prior to the onset of acute weakness. *Id*. Petitioner's treating physicians raised multiple other diagnoses in addition to GBS including TM, paresthesias, myelopathy, and proximal myopathy, but no testing was done. Resp. Ex. C at 1-2; Pet. Ex. 2 at 61-66. Dr. Chaudhry highlighted Dr. Lajara-Nanson's disagreement with petitioner's GBS diagnosis because her CSF testing was normal, she did not have motor weakness or distal sensory loss in the upper and lower limbs, and her reflexes returned, which does not occur with GBS. Resp. Ex. C at 1-2; Pet. Ex. 11 at 3.

Further, based on petitioner's prior medical history, Dr. Chaudhry proposed other possible diagnoses including idiopathic intracranial hypertension to explain her headaches, hormonal irregularities, hair loss, tiredness, and unexplained weight gain, and myopathies associated with cardiac arrhythmias to explain her episodes of syncope in early 2016 that were thought to be vasovagal. Resp. Ex. A at 10. Finally, petitioner was eventually diagnosed with obstructive sleep apnea, which is associated with fatigue, tiredness, and headaches. *Id*. at 11.

[41] Ruts et al., *supra* note 29.
[42] Ruts et al., *supra* note 30.
[43] Lewis, *supra* note 31.

In Dr. Chaudhry's opinion, petitioner's GBS diagnosis is unclear, a host of other possible diagnoses were never explored, and her treater Dr. Lajara-Nanson questioned the GBS diagnosis in May 2018.

Dr. Hoke responded to Dr. Chaudhry's opinions, referencing the medical records as showing a "clear demonstration" of ascending weakness, paresthesia, evolving areflexia, and ataxia, all classic features of GBS. Pet. Ex. 15 at 8. He agreed with Dr. Edgar that the absence of cytoalbuminemic dissociation on CSF testing performed early on in GBS is not unusual and that CSF testing is generally used to rule out infectious etiology, which it did here. *Id*. TM was ruled out based on the MRI results and petitioner lacked bladder and bowel problems. *Id*. Dr. Hoke also referenced literature to show that fatigue, muscle weakness, and paresthesia can persist even in GBS patients who show full recovery on nerve conduction studies. *Id*.; Pet. Ex. 17[44]; Pet. Ex. 18[45]; Pet. Ex. 19[46]; Pet. Ex. 20[47]; Pet. Ex. 21.[48] Dr. Hoke commented on Dr. Chaudhry's reliance on Dr. Lajara-Nanson's opinion, referencing literature which "clearly demonstrate[es] the incorrect rationale" in Dr. Lajara-Nanson's assessment that petitioner could not have had GBS because she had good recovery of her reflexes and strength. Pet. Ex. 15 at 8. Dr. Lajara-Nanson saw petitioner 18 months after the onset of her GBS, and while his assessment may have been reflective of petitioner's condition at that time, it does not detract from her GBS diagnosis in 2016. *Id.*; Pet. Ex. 23 at 2. Further, if she had CIDP as suggested by Dr. Chaudhry, she would not have had a normal examination by Dr. Lajara-Nanson. Pet. Ex. 23 at 2; Pet. Ex. 11.

Dr. Hoke added that Dr. Chaudhry failed to address petitioner's symptoms of flaccid paralysis, progressive loss of reflexes, and sensory and motor deficits that evolved over several days, instead focusing on what he deemed to be "waxing and waning" of petitioner's weakness in the three years after her GBS diagnosis. Pet. Ex. 22 at 1.

Dr. Hoke explained that not all GBS patients present with the same symptoms, and the fact that petitioner did not have an EMG is neither her fault nor does it mean she did not have GBS: "[i]t would have been nice to have it as a confirmatory test but her treating physicians felt that the evidence was sufficient to make a diagnosis of GBS after ruling out other alternative diagnoses." Pet. Ex. 23 at 1-2. Dr. Hoke added that Dr. Chaudhry claimed petitioner had a lack of sensory findings atypical for GBS, but the records show petitioner clearly reported numbness and tingling of her hands and feet to her PCP on December 12, 2016 were indicative of sensory findings. *Id*. at 2; Pet. Ex. 2 at 55.

Dr. Hoke also disagreed with Dr. Chaudhry's suggestion that petitioner had TM. He acknowledged the reference to artifact on the MRI but noted that petitioner never had upper motor neuron signs throughout her hospitalization, which argues against a TM diagnosis. Pet. Ex. 23 at 2; Pet. Ex. 2 at 58. Further, petitioner's rapid loss of reflexes fits the presentation for GBS, not TM or MS. While petitioner had preserved reflexes on December 11, 2016, she was noted to be

[44] Forsberg et al., *supra* note 3.
[45] Garssen et al., *supra* note 18.
[46] Merkies & Kieseier, *supra* note 19.
[47] Rekand et al., *supra* note 6.
[48] Garssen et al., *supra* note 4.

areflexic when she was first examined by Dr. Edgar upon admission to the hospital. Pet. Ex. 23 at 2; Pet. Ex. 4 at 31-32.

Dr. Hoke concluded that although Dr. Chaudhry raised several possible alternative diagnoses including CIDP, TM, and MS, the required testing to advance those diagnoses was never performed. Dr. Chaudhry conceded that petitioner's treating physicians diagnosed her with "probable GBS", and as a specialist in peripheral nerve disorders like GBS, Dr. Hoke believes GBS is the correct diagnosis. Pet. Ex. 23 at 2.

The issue with Dr. Chaudhry's opinions is that while many different diagnoses were considered by petitioner's treating physicians, once the CSF confirmed that there was no infectious process and the MRI was determined to be normal, all of petitioner's physicians agreed that she had GBS with no further testing deemed necessary. IVIG was started, she began to improve, and reached a plateau. She then continued to suffer from what her treating physicians considered to be residual symptoms of her GBS. Dr. Edgar specifically noted that CSF testing done too early, five days after onset in this case, is commonly normal. Petitioner cannot be penalized for additional testing her physicians did not think was necessary based on her clinical presentation and response to treatment. She was diagnosed with GBS by all but one of her treating physicians, and that one did not see her until 18 months after her illness.

Further, CIDP is a slowly progressive demyelinating polyneuropathy, while GBS is an acute monophasic peripheral neuropathy. Between December 11, 2016 and December 14, 2016, petitioner had rapidly developing upper and lower body weakness with numbness, tingling, ataxia, areflexia, facial diplegia, and loss of motor and sensory.

The totality of the evidence does not demonstrate that a more likely diagnosis exists and supports petitioner's diagnosis of GBS, meeting this requirement.

4. Alternative Causation

Dr. Chaudhry argued that even if it were assumed that she did suffer from GBS, evidence of preceding infections may have been the antecedent trigger. The flu vaccine was not likely the cause of her symptoms. Resp. Ex. A at 11.

Dr. Chaudhry argued that GBS is a post-infectious immune disorder, with two thirds of patients having diarrhea or an upper respiratory illness several weeks before the onset of symptoms. Resp. Ex. A at 11; *see* Resp. Ex. A, Tab 1[49]; Resp. Ex. A, Tab 2[50]; Resp. Ex. A, Tab 3.[51] GBS has been associated with C. jejuni infection, found in 25-50% of GBS patients, cytomegalovirus, Epstein-Barr virus, influenza A virus, mycoplasma pneumoniae, Haemophilus influenza, hepatitis E, Zika, and chikungunya. GBS has also been reported following vaccination for rabies and rabies and influenza A. Resp. Ex. A at 11; Resp. Ex. A, Tab 1[52] at 2.

[49] Willison et al., *supra* note 5.
[50] Vriesendorp, *supra* note 8.
[51] Fokke et al., *supra* note 13.
[52] Willison et al., *supra* note 5.

According to Dr. Chaudhry, petitioner had three possible infections prior to her onset of weakness and paresthesias. The first was an upper respiratory infection on October 31, 2016, five to six weeks prior to onset, with sore throat, cough, wheezing, and chest tightness. Resp. Ex. A at 11; Pet. Ex. 2 at 48. The second was another upper respiratory infection on December 7, 2016, three days prior to onset, with head and chest congestion, earaches, nasal drainage, and cough. Resp. Ex. A at 11; Pet. Ex. 2 at 51. Third, bloodwork done on December 13, 2016 was positive for EBVG and EBNUCL AB, though Dr. Chaudhry conceded these findings do not suggest acute infection, but noted that Epstein-Barr Virus is a known antecedent to GBS. Resp. Ex. A at 11; Resp. Ex. C at 4; Pet. Ex. 2 at 74. In Dr. Chaudhry's opinion, if petitioner had GBS, it was caused by the immediate antecedent upper respiratory infection, and Dr. Hoke ignored these infections. Resp. Ex. A at 11-13; Pet. Ex. 2 at 61-66, 68, 82-83, 88-93, 111; Pet. Ex. 4 at 47. Dr. Chaudhry contends that the failure to identify a specific infection does not mean infection was not the cause of petitioner's GBS. Resp. Ex. C at 3.

Dr. Chaudhry argued that if petitioner did have GBS, it was caused by the viral infection that preceded her illness because upper respiratory infections precede 38% of cases of GBS. Resp. Ex. A at 13-14. Therefore, he opined that petitioner's infection(s) were more likely the cause of her neurological symptoms. The literature does indeed provide statistics regarding the association between viral infections, surgery, pregnancy, toxins and GBS. Resp. Ex. C at 3-4; Resp. Ex. C, Tab 3.[53]

Dr. Hoke pointed out that although several medical providers referenced petitioner's upper respiratory infection and sinus congestion "at some point prior to the onset of her GBS", they did not attribute her GBS to those infectious causes. Pet. Ex. 22 at 1. The medical records do not show that any of her treating physicians believed any of the common infections responsible for GBS were responsible for petitioner's GBS. *Id*. Dr. Hoke contends that the history of infection noted in the record was simply a reference to temporal events around the time of her onset of GBS. Pet. Ex. 23 at 1. There was no record of any specific infection and Dr. Chaudhry agreed that blood work did not show any infection believed to cause GBS. Further, the exclusionary criteria for a Table injury "makes no reference to ill-defined generalized infectious processes" such as a sinus infection, congestion, or upper respiratory infection. Dr. Hoke maintained his opinion that petitioner suffered a Table injury. Pet. Ex. 22 at 2.

The Federal Circuit has held that:

> [T]he bare statistical fact that there are more reported cases of viral encephalopathies than there are reported cases of DTP encephalopathies is not evidence that in a particular case an encephalopathy following a DTP vaccination was in fact caused by a viral infection present in the child and not caused by the DTP vaccine.

Knudsen by Knudsen v. Sec'y of Dep't of Health & Human Servs., 35 F.3d 543, 550 (Fed. Cir. 1994). The Federal Circuit repeated this view in *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351 (Fed. Cir. 2019), holding that the special master impermissibly found based on statistical

[53] J.B. Winer et al., *A prospective study of acute idiopathic neuropathy. II. Antecedent events*, 51 J. NEUROLOGY, NEUROSURGERY, & PSYCHIATRY 613 (1988), filed as "Resp. Ex. C, Tab 3."

evidence alone that the child had a defective brainstem, making him vulnerable to vaccines. Petitioner rightfully argues that *Knudsen* makes clear that it is legally irrelevant that infections may be responsible for more cases of GBS than the influenza vaccine, and it is therefore "prohibited" for the special master to consider statistical evidence "based strictly on probabilities" without more to defeat a Table claim. Motion at 4. Thus, without more, statistical analyses such as that employed by Dr. Chaudhry are legally irrelevant to determining vaccine causation. *Id*. at 4-5.

Further, the Vaccine Injury Table QAIs contain exclusionary criteria for GBS, but infections are not one of them. 42 C.F.R. § 100.3(c)(15)(vi). Other than his reliance on the literature that infections are a known cause of GBS, Dr. Chaudhry provided no evidence to show that an infection caused petitioner's GBS in this case. Simply showing that an infection can cause GBS does not satisfy respondent's burden; he must also show by preponderant evidence that the infection did cause petitioner's GBS. Respondent failed to do so.

Petitioner has presented sufficient evidence that she has satisfied the requirements of a Table GBS claim.

V. Conclusion

Based on the record as a whole and for the reasons discussed above, I find there is preponderant evidence that petitioner meets the Table criteria for GBS. Therefore, petitioner's Motion for Ruling on the Record is granted, and petitioner is entitled to compensation. Accordingly, this matter shall proceed to damages.

IT IS SO ORDERED.

s/ Mindy Michaels Roth
Mindy Michaels Roth
Special Master